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Beersma, Domien G.M.; Dijk, Derk-Jan

Published in:
Journal of Sleep Research

DOI:
[10.1111/j.1365-2869.1992.tb00039.x](https://doi.org/10.1111/j.1365-2869.1992.tb00039.x)

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Document Version
Publisher's PDF, also known as Version of record

Publication date:
1992

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

Beersma, D. G. M., & Dijk, D.-J. (1992). Selective SWS suppression does not affect the time course of core body temperature in men. *Journal of Sleep Research*, 1(3), 201-204. <https://doi.org/10.1111/j.1365-2869.1992.tb00039.x>

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Selective SWS suppression does not affect the time course of core body temperature in men

DOMIEN G. M. BEERSMA¹ and DERK-JAN DIJK²

¹Department of Biological Psychiatry, University of Groningen, Groningen, The Netherlands; ²Laboratory for Circadian and Sleep Disorders Medicine, Harvard Medical School, Brigham and Women's Hospital, Boston USA

Accepted in revised form 1 May 1992; received 10 February 1992

SUMMARY In eight healthy middle-aged men, sleep and core body temperature were recorded under baseline conditions, during all-night SWS suppression by acoustic stimulation, and during undisturbed recovery sleep. SWS suppression resulted in a marked reduction of sleep stages 3 and 4 but did not affect the time course of core body temperature. These data suggest that sleep stages 3 and 4 of nonREM sleep (i.e. SWS) do not play a major role in the regulation of core body temperature in humans.

KEYWORDS body temperature, deprivation, human, slow-wave sleep

INTRODUCTION

Glotzbach and Heller (1976) showed in kangaroo rats that the hypothalamic temperature at which a metabolic heat production response occurs is lowered during slow-wave sleep (SWS; which was all of nonREM sleep and not a substage of nonREM sleep as in humans). Furthermore, compared to wakefulness, the proportionality constant relating heat production to hypothalamic temperature was lowered. These data led to the hypothesis that SWS has an energy conservation or thermoregulatory function and inspired research on the relationship between SWS and temperature regulation in humans. In humans, core body temperature falls at the beginning of a sleep episode where SWS (i.e. Stages 3 and 4 of nonREM sleep) is most prominent. Both the drop in core body temperature and the amount of SWS at the beginning of a sleep episode are to a large extent independent of the circadian phase, at which sleep is initiated (Åkerstedt and Gillberg 1981; Gillberg and Åkerstedt 1982; Dijk *et al.* 1990). Induced elevations of core body temperature by 2°C or more (Horne and Staff 1983; Horne and Reid 1985; Shapiro *et al.* 1989) have been shown to increase SWS in the subsequent sleep episode, provided that this heat stress does not occur too close to sleep onset (Bunnell *et al.* 1988). SWS can be induced to occur in the second half of a sleep episode by elevating core body temperature in the middle of the night (Bunnell and Horvath, 1985). Furthermore a positive correlation between core body temperature at the beginning of sleep and

subsequent SWS has been reported (Berger *et al.* 1988). All these data are in accordance with an association between temperature regulation or thermal load and subsequent SWS (McGinty and Szymusiak 1990). The question whether SWS serves a down regulation of body temperature, and hence whether changes in SWS do also result in changes in the time course of body temperature has, to our knowledge, not been addressed. The electrophysiological signs of SWS, delta waves, can be easily suppressed by acoustic stimulation without inducing a substantial increase of intermittent wakefulness. Because after termination of such a selective SWS suppression a compensatory increase in SWS and slow-wave activity (SWA) is observed (Dijk *et al.* 1987; Dijk and Beersma 1989; Gillberg *et al.* 1991), this manipulation not only affects the electrophysiological correlates of SWS but also some of the regulatory processes underlying SWS. Therefore, this paradigm is suited to investigate the influence of SWS on the course of body temperature.

METHODS

Eight middle-aged male subjects (mean age 49.8 years, range 42–56) were selected on the basis of a questionnaire. They all reported good health and were free of sleep complaints, as assessed by a general sleep complaints scale. They gave written informed consent and were instructed to refrain from alcohol or drugs for the duration of the experiment, not to take naps and to avoid irregular activities or heavy exercise. Core body temperature was recorded via a rectal probe on a Vitalog throughout the study. Subjects slept in the laboratory at their habitual bedtimes. After an adaptation night, baseline sleep was recorded, the results of

Correspondence: Dr D. G. M. Beersma, Department of Biological Psychiatry, Oostersingel 59, 9713 EZ Groningen, The Netherlands. Fax #31/50-696727

which have been reported before (Dijk *et al.* 1989). On the next evening SWS was suppressed by acoustic stimulation for the duration of the entire sleep episode. Attempts were made to induce as little wakefulness as possible by each time increasing the loudness of the acoustic stimuli until a change in the EEG was observed, after which loudness was immediately attenuated again. Recovery sleep was recorded on the next evening.

EEGs were recorded from C3-A2 and C4-A1. The EEG signals were low pass filtered at 25 Hz (24 dB/octave) and subsequently digitized at a sampling rate of 64 Hz for spectral analysis. All EEG records were scored according to established criteria. Ambient temperature was regulated through the hospital heating system. As a result ambient temperature was about 20°C. Bedding and clothing was the same for the three conditions.

Temperature data were analysed as follows. Data were obtained at a resolution of 0.1°C and a sampling rate of 4 samples per min. The time series of data was compressed to 1-min values by averaging over 4 consecutive values. For each 1-min interval medians were calculated over subjects. In addition, differences between conditions were calculated by subtracting the 1-min values in baseline from the values at the same clocktime in the SWS suppression night; and by subtracting the values in the SWS suppression night from the values obtained during recovery.

For the statistical analysis, baseline nights and SWS suppression nights were compared per 1-min interval as well as per 1 h interval by means of the Wilcoxon matched-pairs signed rank test.

RESULTS

The SWS suppression procedure resulted in a dramatic reduction of SWS and a significant increase in wakefulness (Table 1). The latency from sleep onset (first occurrence of Stage 2) to REM sleep increased. Total REM sleep was not affected when compared to baseline; however, it was significantly less than during recovery sleep. Figure 1 illustrates the time course of SWA, the sleep stages, and core body temperature on the baseline night, the SWS suppression night and the recovery night for one subject.

Although SWS suppression was quite successful in that it reduced SWS, it did not affect the time course of core body temperature. In Fig. 2 the time course of body temperature is presented for the group of subjects for all three conditions. In addition, the course of body temperature in baseline is subtracted from the time course during SWS suppression and the course during SWS suppression is subtracted from the course during recovery. There were no 1-min intervals (nor 1-h intervals) in which core body temperature during SWS suppression was significantly higher than during baseline. The same holds for the comparison between SWS suppression and recovery. Actually, in some intervals a non-significant trend towards lower values was present in the SWS suppression condition.

DISCUSSION

The present data show that a 58% reduction of SWS does not induce any significant change in the time course of core body temperature. Since SWS suppression results in a rebound of SWS after termination of the deprivation (Dijk *et al.* 1987), this manipulation does not only interfere with the electrophysiological correlates of SWS but also with at least some of its regulatory processes. SWS suppression as applied in the present experiment did induce an increase in intermittent wakefulness, accompanied by a relatively smaller reduction in REM sleep. If anything, one would expect that an increase in intermittent wakefulness would lead to an increase in core body temperature, possibly partially compensated by the reduction in REM sleep. Since there are no signs of an increase in body temperature in the data, it can only be concluded that SWS suppression does not lead to an increase in body temperature.

A dissociation between the time course of SWS/SWA and core body temperature has been observed in two other recent experiments. During extended sleep episodes of up to 15 h, the early morning/afternoon rise in core body temperature was not associated with an increase in SWS/SWA (Dijk *et al.* 1991b). Exposure to bright light prior to sleep, which resulted in an elevation of core body temperature of 0.3°C that persisted during the first 4 h of sleep, did not affect SWS (Dijk *et al.* 1991a) or SWA. These

	Baseline	SWS supp.	<i>P</i> *	Recovery	<i>P</i> #
sleep onset	00.05 ± 24	00.14 ± 64		23.55 ± 43	
sleep end	07.08 ± 32	07.23 ± 38		07.13 ± 86	
REM latency	81.5 ± 15	114.4 ± 26	0.042	70.0 ± 12	0.021
First 6 h after sleep onset:					
SWS	63.9 ± 52	26.4 ± 38	0.025	81.0 ± 56	0.014
REMS	62.7 ± 14	45.6 ± 22		64.1 ± 21	0.021
Intermittent waking	10.6 ± 9	50.1 ± 39	0.014	15.2 ± 19	0.014

Table 1 Sleep measures during baseline, SWS suppression, and recovery nights in eight middle-aged male subjects.

Sleep onset: clock time of first occurrence of Stage 2, Sleep end: clocktime of last occurrence of Stage 2, REM latency: time from sleep onset until first occurrence of REM sleep, * indicates significant differences between baseline and SWS suppression (Wilcoxon matched pairs, 2-sided), # indicates significant differences between recovery and SWS suppression. Values are in minutes.

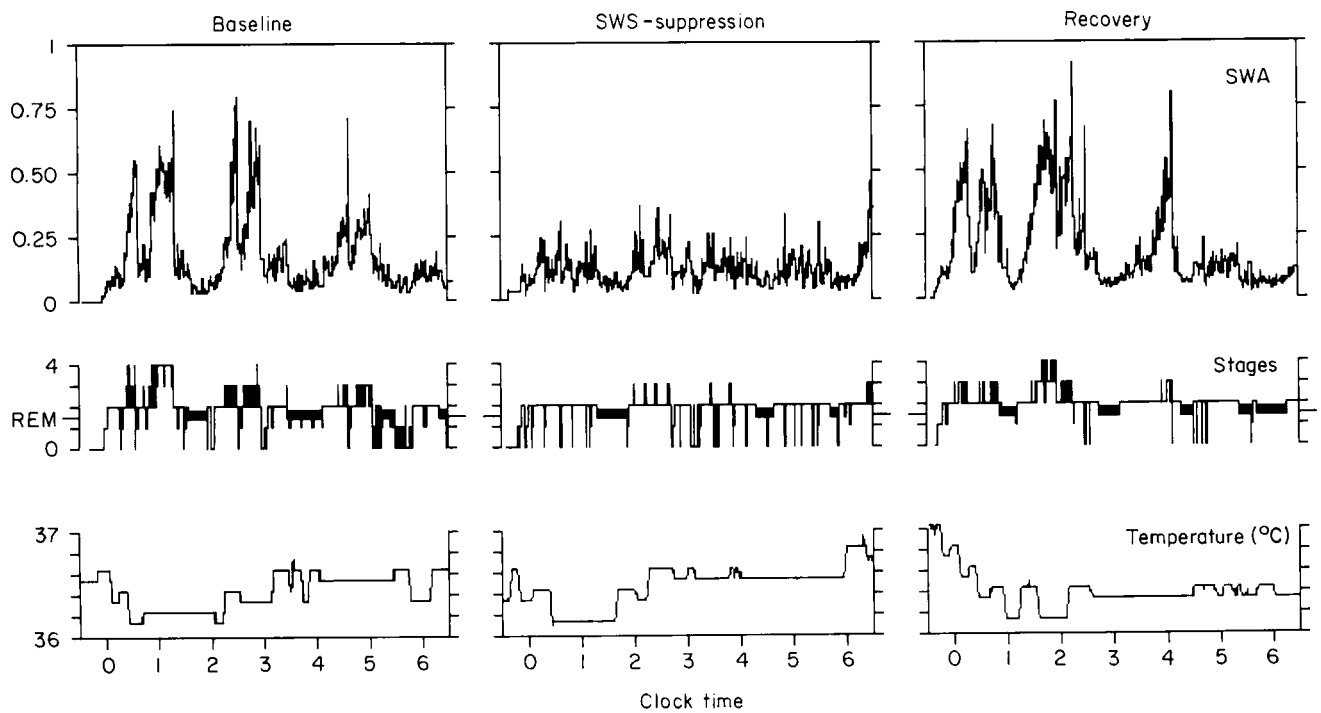


Figure 1. Time course of SWA (i.e. power density 1.0–4.75 Hz), sleep stages and core body temperature during baseline (Left panel), during SWS suppression (Middle panel), and during recovery sleep (Right panel) in one subject. SWA is expressed in relative units. Sleep stage 0 corresponds to wakefulness.

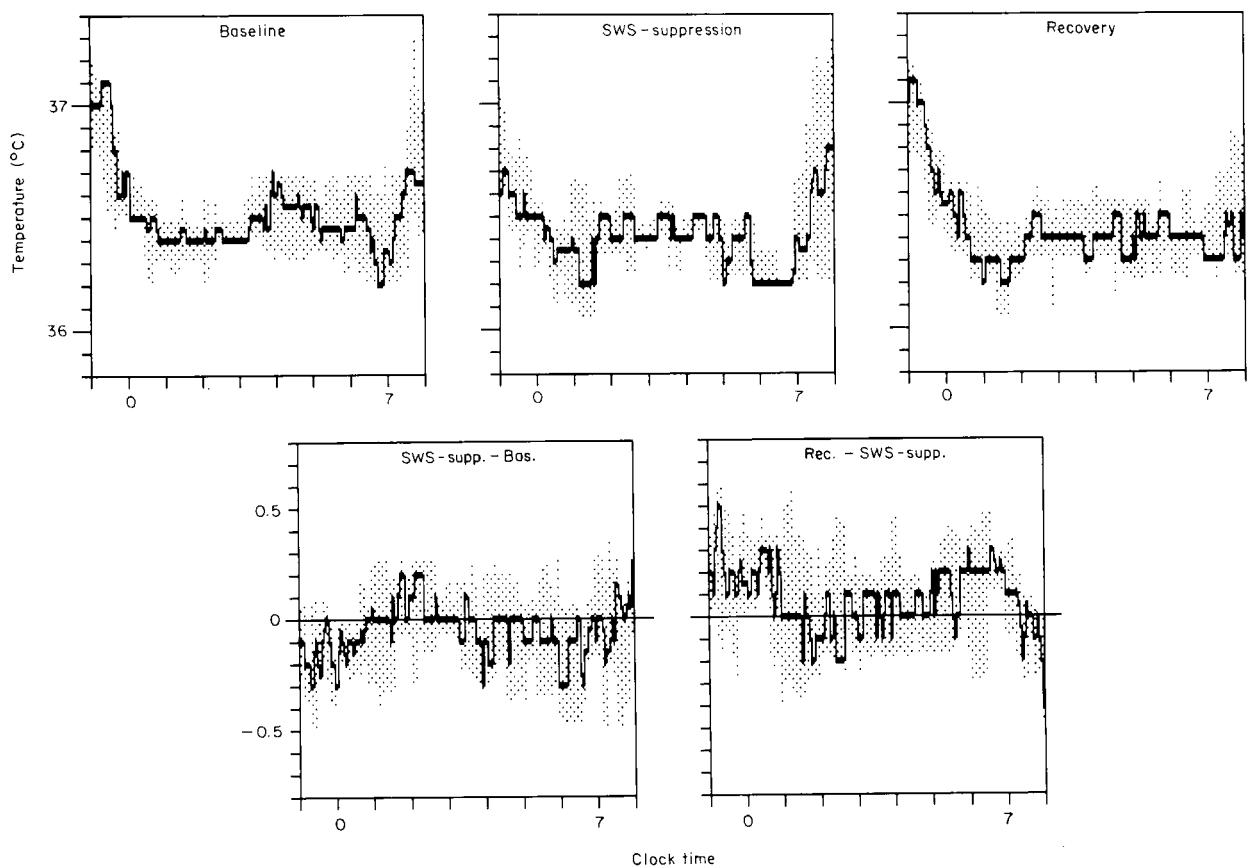


Figure 2. The course of body temperature during baseline, SWS suppression and recovery nights as a function of clocktime in eight middle-aged male subjects (upper graphs). The difference in core body temperature values between successive conditions is presented in the lower graphs. Median values and quartile ranges are presented. The common sleep interval is between 24.00 hours and 07.00 hours.

data indicate that the relationship between SWS/SWA and core body temperature is not as close as sometimes assumed (Sewitch 1987; McGinty and Szymusiak 1990).

It may be argued that SWS suppression perhaps had functionally relevant repercussions on the time course of brain temperature, which are not reliably reflected by core body temperature measurements. In humans, dissociations between core body temperature and tympanic temperature have indeed been reported (Palca *et al.* 1987). Although we cannot rule out this possibility, evidence against a close relation between brain temperature and SWA has been obtained in the rat. In their analysis of the correlations between sleep stages, SWA and cortical temperature, Franken *et al.* (1991) obtained a very strong correlation between the duration of nonREM sleep and brain temperature. However, they failed to find a significant correlation between SWA in nonREMS and brain temperature, indicating a dissociation between the regulation of brain temperature and SWA.

In conclusion it seems that although elevation of core body temperature prior to sleep by 2°C or more influences the duration of the sleep stages 3 and 4, the manipulation of these sleep stages, in turn, does not influence the time course of body temperature.

ACKNOWLEDGEMENT

The research assistance of Gerda Bloem is gratefully acknowledged.

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